SPEECH DISORDERS AND THEIR TREATMENT*

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IN order to understand speech disorders, one must know something of the development of speech in man and his ancestors. In phylogeny there is evident a close relationship between the development of binocular vision, manual skill, and finally a leading hand and leading cerebral hemisphere. When man's shrew-like ancestors took to trees and left behind them quadrupedal locomotion, their fore-limbs were emancipated to become hands, and the possibility of dexterity developed. Other vertebrates specialize and reach narrow superiority by unique development of one type of sense organ. Man excels because of lack of specialization. His speciality is a leading hemisphere, evenly developed in the reception and elaboration of all types of sensory impression. It is this even development that allows for association between the different sensory receiving stations; and it is the development of the mechanism of association that has made intellectual behavior possible. The dominance of the left hemisphere is marked in right-handed people, who constitute about 75 per cent of the population. The functions are probably not confined to the left hemisphere; the dominance is relative. Recent studies have shown that even speech is subserved in a rudimentary way in the right hemisphere of right-handed persons. With a leading hemisphere in which there is a predominant centering of manual skill (eupraxia), symbolic understanding (eugnosia), and language, there is a great need of many associative tracts. The function of language needs both eupraxia and eugnosia, in fact, each needs the other two for effective behavior. Nevertheless it can be taken as a proposition that language is the most highly integrated of man's functions.[1,2,3]

In the central nervous system of man, speech is integrated at five important levels; these are not exact, and to some extent overlap but for purposes of exposition Fig. 1 shows the main centers involved. The

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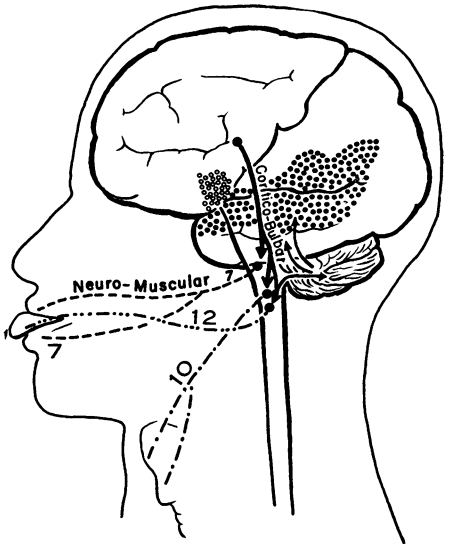

V—Social, Personal, Emotional, etc.

IV—Symbolic, Inter-personal

III—Cerebellar

II—Cortico-bulbar

I—Neuro-muscular

Fig. 1

broken lines show the lowest level, the neuron from a nucleus in the medulla (seventh, tenth or twelfth) to a peripheral organ of speech, lips, tongue or larynx. This is level I, the neuromuscular neurons and the final common paths over which all nerve impulses concerned with speech must pass. Level II is the neuron arising in the motor cortex and ending in the medulla around the bulbar nuclei (seventh, tenth and twelfth), its fibers pass down the corona radiata, through the internal capsule to the peduncles and medulla. This, too, is a common pathway for all the complex speech integrations of the higher levels. It is closely correlated for motor speech production with the cerebellar paths (Level III) which run up from the medulla to the cerebellar cortex, thence to cerebellar efferent nuclei and upward to mid-brain, thalamus and motor cortex. This makes a loop and supplies the mechanism of coördination so greatly needed in the control of speech. These three levels can be considered together as a rather straightforward motor mechanism.

Moving up to Level IV is a big step, for symbolization and meaning come in here. The anatomical localization is cortical with intricate association fibers between the different parts of the speech areas (dotted on cortex of Fig. 1). The circular stippling indicates the motor speech area (Broca), the black stipples show the auditory and visual areas of langu-

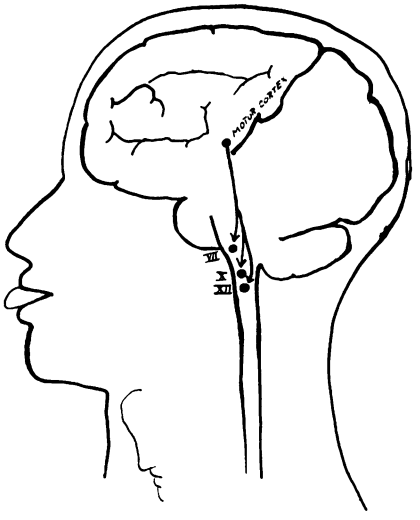

Fig. 2

LEVEL II—CORTICO-BULBAR

Common Lesions: Syphilis (G.P.I.), Cerebral Arteriosclerosis

Clinical Results: Dysarthria, Weakness, Paresis or Partial Paralysis

Treatment: Antiluetic or none

age functions and their subsidiaries. These areas are well developed in only one hemisphere, the "leading hemisphere," usually the left. The degree of dominance is important.

Level V is the most highly integrated and least localizable. It is associated with the great association area of the frontal and parietal lobes. It brings in memories, individual life experiences and the impact of one person upon another including the varied and important emotional reactions expressed by glands, smooth muscle and skeletal muscle. The visceral expressions are mediated by the hypothalamus and the autonomic system; the overt behavior responses are expressed largely by skeletal muscles as behavior. Although both may *express* emotions, they are not the *sources* of emotion, which must be looked upon as largely cortical, individual and extremely complex.

Taking up each level in turn in regard to (a) common lesions, (b) clinical results of these lesions, and (c) treatment, one finds little of interest in level I. The lesions are usually the obvious developmental defects of the end organs treated by surgery, e.g., hare-lip and cleft palate. Tying down of the tongue by a short frenulum is emphasized by some but is actually a rarity. Trauma and neuritis may paralyze the nerves. Surgical anastomosis may be necessary. Poliomyelitis or more rare degenerative diseases may cause nuclear lesions and, hence, bulbar

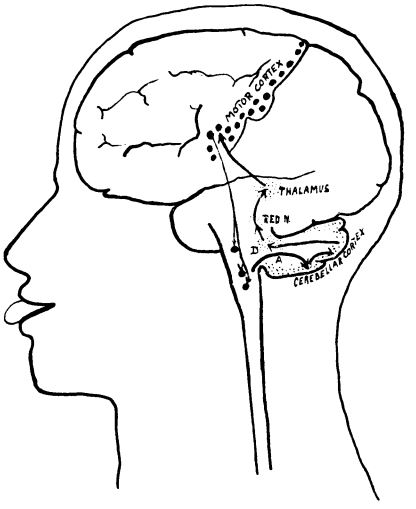

Fig. 3

LEVEL III—CEREBELLAR

Common Lesions: Multiple Sclerosis, Tumor, Encephalitis

Clinical Results: Incoordinate Speech, Scanning, Monotonous, Flat, Explosive, Staccato, etc.

Treatment: Possible Removal of Tumor; Usually None

palsy. Treatment is unsatisfactory; excessive doses of vitamins may be tried.

At Level II the cortico-bulbar neurons are found to be frequently affected by syphilis, as in dementia paralytica ("G.P.I.") and by cortical softenings from arteriosclerosis. Other pathological processes, such as tumor or encephalitis lethargica, rarely damage these neurons in a selective way. The result of injury to the nerve cells of the motor area which innervate bulbar speech mechanism is weakness, partial paralysis. It is partial because the innervation is bilateral, neurons from one motor area going to the nuclei on both sides of the medulla, although, predominantly to the opposite side. The clinical symptom produced by this weakness is *dysarthria* or slurred speech. The classical example is the patient with "G.P.I." who cannot enunciate "Methodist episcopal" but says something like "Methyst epistople." The treatment of this sort of bad enunciation is unsatisfactory. Antiluetic treatment sometimes brings back paretics' poor speech. Usually little or nothing can be done. The slovenly speech of uneducated persons must be ruled out by repeated trials of different test phrases and attention to other clinical symptoms.

The third level (III in Fig. 1) is elaborated in Fig. 3 to show more details of the cerebellar coördinating mechanism. Nerve impulses from cells in the medulla enter the cerebellum over afferent tracts (A) bringing in proprioceptive information, data as to what the muscles are doing.

This is spread through the cerebellar cortex by the plentiful association fibers. Impulses then leave the cortex of the cerebellum and go to dentate nucleus (D) whence they are relayed upward through the red nucleus to the thalamus. From here another neuron takes them to the motor cortex of the cerebrum where they have their effect on the motor impulses sent out from headquarters. Impulses from the roof nuclei of the cerebellum and from the red nucleus probably go directly down to brainstem and spinal centers to effect further motor control. Thus a loop or series of loops, is formed for making the delicate motor adjustments of speech all work together. The mechanism is for coördination or synergia.

Lesions anywhere along this pathway will cause trouble. The commonest to disrupt smooth speech are those of multiple sclerosis and epidemic encephalitis; tumors and softenings may also do the same. The clinical results are incoördination (asynergia) of speech, shown in the monotonous speech of Parkinson's disease (where the lesion is probably up near the thalamus), in the scanning speech of multiple sclerosis, and in the various explosive and staccato forms of speech found in encephalitis. Except for the rare removal of tumors, treatment is of no avail.

Level IV is divided in two parts (a) and (b) because (a) takes in the functions of the dominant, leading hemisphere, those functions which are largely absent in the minor hemisphere, while (b) is the relationship between the leading and minor hemispheres. It deals with the degree of dominance of one over the other.

Level IVa (Fig. 4) is concerned with symbolism. Symbols must be sensed, recognized and understood as meaning something. The senses important to language are vision and hearing, each has three levels of integration. Visual reception is localized in the occipital pole and visual recognition in the stippled area just anterior to this. A lesion at the first level causes blindness; at the second, loss of recognition of perceived objects, in other words, visual agnosia. A lesion farther forward, but still behind the angular gyrus (LEX Fig. 4) causes visual disorientation. The patient cannot remember the plan of a house, or he gets lost, although he may recognize the street or house he is seeing at the moment, because he cannot bring back series of pictures.

The auditory sense has three similar levels in the temporal lobe: auditory reception (AUD. REC., Fig. 4), auditory recognition of noises

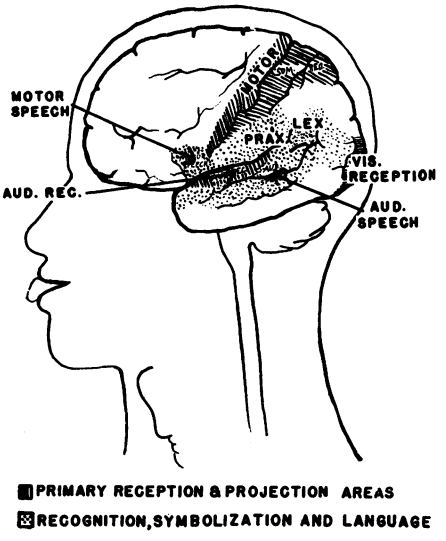

Fig. 4

LEVEL IVa—SYMBOLIC

Common Lesions: Arteriosclerotic Softening, Tumor and Trauma

Clinical Results: Deafness and Blindness, Apraxia and Motor Aphasia, Visual and Vis. Verbal Agnosia, Auditory and Aud. Verbal Agnosia, Agraphia, Amusia, Acalculia, etc., Semantic and Amnesic Aphasia

LEVEL IVb—LATERAL DOMINANCE

Common Lesion: Unknown, Inherited

Clinical Result: Slow Reading, Reversals, etc., Stammering, Ambidexterity

Treatment: Retraining by Reading, Speaking, Writing, etc. (Orton)

and auditory recognition of symbols (words). Since the innervation of the temporal lobe is bilateral, a single cerebral lesion does not necessarily cause complete agnosia, and only bilateral lesions can cause deafness. Acoustic agnosia for sounds is not known to occur without some acoustic verbal agnosia, but in many cases of the latter the recognition for sounds is retained. Thus it is obvious that the acoustic mechanism is more variable and less unilaterally dominant than the visual mechanism. Recognition of learned symbols for words seen is in the angular gyrus, (LEX Fig. 4), and a lesion here causes visual verbal agnosia. The patient cannot read, he has "alexia" or "word blindness," as opposed to the "word deafness" resulting from a lesion in the temporal lobe.

Motor dysphasia* is equally complex and depends on an understanding of the fact that no learned motor skill can be practiced without an ideational plan. This is a psychic elaboration necessary as a precursor to the carrying out of any complex motor act. This ideational planning is called eupraxia; the normal person knows how to do a thing quickly and almost automatically when requested. A defect of such performance in response to command is apraxia. It is a symptom of injury to the sensorimotor elaboration areas of the cortex, corresponding to

* Dysphasia, meaning disturbed language, is more accurate in most cases than aphasia, which means lack of language.

agnosia from a lesion in the more strictly sensory area. In relation to most performances eupraxia is bilateral, the acts of one hand, for example, being planned in the opposite precentral and supramarginal gyri (between PRAX and LEX, Fig. 4). Lesions of these areas or their fiber connections may cause dyspraxia or apraxia.

Apraxia of the organs of speech (tongue, lips, larynx) causes "motor aphasia"; the patient loses the memory of how to make the movements to articulate words; he may know just what he wants to say, but he cannot get the plan of the word into his mind. A lesion of Broca's area (MOTOR SPEECH, Fig. 4) causes this symptom. As a rule only propositional speech is lost when the major motor speech area is injured. This is because the function of language is largely, but not entirely, concentrated in the leading hemisphere; a primitive sort of speech (emotional, expletive, habitual) is subserved in the homologous areas of the minor hemisphere. Individual variation in relation to the degree of handedness is important.

The angular gyrus has the function of symbolic visual recognition, i.e., understanding letters and words by sight. Because words were first learned by hearing, however, this area cannot function if cut off from the temporal lobe. During the process of learning to read, words are sounded out or told to one. Thus the portion of the temporal lobe lying behind "Aud. Speech" (Fig. 4) is of great importance. A lesion here may cause as much loss of reading ability (alexia) as a lesion directly in the angular gyrus. Moreover, because language is learned by associating vision and hearing with objects and symbols, this area is particularly important, Nielsen[4] calls it "the language formation area." Take, for example, the case of objective nouns, the simplest and commonest words used. They are names, usually of objects seen. So memories must be stored in the posterior temporal lobe correlating both visual and auditory meanings. That this is true is shown by cases of temporal lobectomy in which the language formation area of the leading hemisphere is removed. The result is amnesic aphasia. The patient has lost the storage mechanism for nouns; his "naming center" is gone. When shown a pen, he uses circumlocution and may reply "It's to write with" but cannot say "pen."

If one takes these main symptoms (which are well explained physiologically), agnosia, motor aphasia and amnesic aphasia, and adds the obvious complications, it is seen that many special sorts of aphasia are

possible. But by sticking to the principles laid down, these strange phenomena can largely be explained and localized. For example, one can hear music as well as words; therefore "amusia" is found. One can learn several languages and have marked aphasia in one but little in another. Symbols used for mathematics are different from letters; so one may have agnosia for figures. In fact, loss of the ability to calculate is found after lesions in various areas. Semantic aphasia should be mentioned because it is the commonest of all aphasias and is not localizable. It consists of a quantitative reduction in the capacity for and comprehension of speech. The victim has a little of all language functions left but cannot put sentences together or express any complex idea. This phenomenon is present in persons with diffuse cerebral lesions, commonly senile, in toxic patients and in normal but excessively fatigued persons. In fact, almost all patients with aphasia have an element of the semantic form, and careful examination will usually show some mental loss. Semantic aphasia is a form of dementia.

The lesions causing dysphasia and dyspraxia are varied, the commonest being trauma, tumor and softening. Treatment is often not pushed as much as it ought to be. When the first shock of the lesion or operation is over, and the patient is comfortable, re-education should be started. First comes accurate diagnosis,* because the physician must first find out what parts of the language mechanism remain intact in order that he may work on and through these normal or less injured parts. Of course it is equally important to learn how much language the patient had before his cerebral lesion. Was he a laborer with a vocabulary of only a couple of thousand words in one language? Was he a waiter with glib use of five languages, or perhaps a college professor with many thousands of words in two or three languages? The diagnosis and prognosis differ in each case.

Having discovered what sort of a person one has to deal with and what is left of the cerebral speech mechanism, one can outline treatment. If the aphasia is complete there is little to do but to stimulate the patient to more activity and more contact with environment by moving about and occupational therapy. As speech begins to return a place may be found for the entering wedge of treatment. If the case is one of dysphasia ingenuity must be used to make up exercises which will be understood and will re-educate injured cerebral areas or educate the

* The best short method is that of Cheser, E. C., *Bull. Neurol. Inst. New York*, 1937, *6:* 134.

unused area in the minor hemisphere. The evidence is fairly good that the latter is what probably takes place.

For example a patient with visual agnosia cannot recognize objects by sight; he must be shown objects and then be given the cue as to what they are by touch and sound. He looks at the object, feels it and hears the name all at once, and repeatedly. If he has a motor dysphasia with agraphia he is taught to write with his left hand from copy and from dictation. Also he practices speaking words by naming objects while writing the name. The training work is best done by non-medical teachers who have learned the methods in general and who have had from the neurologist special instruction for each case. Goldstein[5] in his recent book shows how varied are the problems and how ingenious the physician must be in planning the therapeutic attack. It is certain, however, that much can be done, especially in young and middle aged people. The teacher must be patient, go slowly and not work long at any one session. A few minutes often tires the patient.

There will be many cases of dysphasia among the many soldiers with head wounds. The specialists in this line of treatment are few and they should begin now to train assistants and teachers to carry out the re-educational procedures. Moreover, many old people with vascular lesions of the brain would be benefited by these methods of treatment, yet most of them are looked upon as hopeless.

Level IVb is actually the same level of structure and integration as IVa, but it deals not only with the function of the dominant hemisphere, but with the relative dominance of the two. Which is dominant and to what extent? In other words there is no 100 per cent use of one and 0 of the other; most persons are very largely right- or left-handed, but 10 or 15 per cent of the population are ambidextrous, have no clear dominance and thus have a lack of leadership in the initiation of speech, writing and reading. There is a mixed lead. One result may be hesitant speech and stuttering; another may be reversed symbols and "strephosymbolia" as described by Orton[6] where letter symbols are confused when similarly formed but differently oriented, as "d" and "b," or words such as "was" and "saw." The left-handed child would like to read from right to left but unfortunately in a right-handed world the books are not printed that way. Uncertainty of dominance is much more common in infants than later in life when habit has settled the question. In other words a *tendency* to right or left handedness is inher-

ited and is only consolidated by habitual use of one side as a leader. In spite of the fact that handedness is inherited, and therefore dependent upon the structure of the genes, no difference can be seen grossly or microscopically between the right and left hemispheres of the brain.

Physiologically it is obvious that one hemisphere leads in normal persons, but lack of clear dominance occurs often. Clinical evidence is impressive that this mechanism of "mixed leads" causes speech and reading defects. Final proof seems to have come from the laboratory, where Lindsley[7] made electroencephalograms of normal people and stammerers during speech. In normals the alpha rhythm of the brain waves was usually synchronous and smooth. In the stammerers the waves in the tracings from the two hemispheres were frequently out of phase and often obliterated, especially when blocking in speech was observed clinically.

Orton's method of training,[6] aimed at making the patient fix his lead in a chosen hemisphere, is an advance in therapy. One must not, however, think that every ambidextrous child will have difficulty in symbolization; perhaps 10 per cent of the population is somewhat ambidextrous and only about one per cent have difficulty. It is probable that ordinary childhood development takes care of most of the cases; habit and use cure the defect; the child "outgrows" his slight disorder. In the cases where the trouble becomes worse and lasts into adolescence and adult life, to become a real disability, one must look for additional causal factors (see level V).

From the standpoint of treatment, it would seem wise to let a left-handed child work out his own salvation. Do not force him to be right-handed in any way; the world will do that gradually because it is a right-handed world the child is born into. The ambidextrous child needs more care; probably he will choose his lead at an early age and follow it, but if reading or speech difficulty develops, he will need visual, auditory and proprioceptive training along Orton's lines to make one hemisphere (preferably the left) clearly dominant. The great desideratum is to accomplish the training with as little emotional stress as possible. Let it be simple and matter of fact. Rather than make it seem too important to the child, give it up, keep the home placid, and wait till the child wants help.

At level V is the highest cerebral complexity of integration. Here associations are somehow stored and conditioned, so that they are indi-

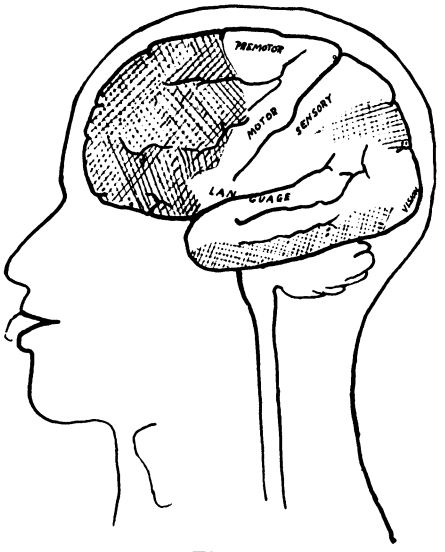

Fig. 5

LEVEL V—SOCIAL, PERSONAL, EMOTIONAL

Common Lesions: None, or Hereditary

Clinical Results: Stammering, Stuttering, Anxiety, Psychoneurosis

Treatment: Ease Tension, Get Relaxation, Restore Confidence—suggestion, group work, accomplishment; Psychiatric talks, Psychoanalysis, Social Service

vidual, depend on past experience and when summed up constitute personality. The areas involved are shown in Fig. 5. These are the obverse of Fig. 4, which maps out all the specialized areas. What is left over is shaded in Fig. 5, and represents what one supposes, on fairly good evidence, to be the association areas, practically blank in infancy and loaded with personal experience in adulthood. These are the parts of the brain that permit the effect of one person upon another to register and effect character. This associative mechanism allows socialization to take place.[2] When the environment is difficult it is this level of cerebral function that is first affected.

When put under unusual emotional stress, almost anyone stammers. Stammerers are people who habitually hesitate and stick in their speech under stress, but who can talk or read with little hesitation when alone and relaxed. The trouble does not seem to be with the organs of speech, they function well enough when the patient is alone; the essential trouble seems to be in the patient's social relations. He is "shy," cannot "put it over," is afraid to meet people, has varied anxieties and especially has become fearful that he will stammer if called on to speak. Certain sounds are especially difficult, but these vary from month to month and year to year, so it is probable that fear of a certain letter is the cause of the sticking, rather than the sound-formation itself. Any spring board

to start the speech and keep it going, like talking in unison or singing, will get rid of the stammer. Putting the patient on the spot by asking him a direct specific question will bring out the blocking in the speech.

For all these reasons stammering is thought to be a psychoneurosis and there is no doubt of the importance of neurosis as one of the causative agents. Everybody probably has stammered at times. It is the child who connects a *fear* that he will stick next time he tries to speak, who perpetuates the stammering into an anxiety state that may last for years. More complicated neurotic mechanisms are usually behind the fear about speech; the child is neurotically anxious and uses speech as the symptom rather than vomiting, bed-wetting, food fussiness or one of the many other possible neurotic expressions.

Experience in treating stammerers has shown that working on the neurosis and paying no attention to the speech will sometimes cure the stammering. Certainly the results of this procedure are better than giving lessons in elocution, breathing, etc. (which almost always make the patient worse) and better than the trick training methods of some of the stammering schools. The tricks of speech learned often help at first because they are taught with much positive suggestion and confidence is gained by the patient.

Probably the best methods are those that combine psychotherapy, socialization and speech exercise. Blanton[8] has written a good book on this subject. Successful schools that do this well are run by Dr. Green in New York and Mr. Martin at Bristol, R. I. The patients must learn to relax, to speak without spasm, to literally "take it easy." Nothing helps this more than good suggestions, properly and repeatedly employed, and success. The success is the most important thing, it allays fear and builds confidence. Speaking in unison, singing, helping each other, all help the patient to extravert himself and shed his anxiety.

Sometimes one line of treatment alone will effect a cure. Jacobson[9] in Chicago has had success with relaxation exercises alone. A few instances are known where psychoanalysis worked. Hypnosis has cured a few. But these phenomena must be looked at as examples of taking a few straws from the camel's back. If one wants to be sure to help, it is far better to use several methods of attack at the same time—directed against the *fear*, the *spasm*, and the social withdrawal. Inheritance is an important causal factor but nothing can be done about it. For the patient it is best forgotten; for the therapist, it should be constantly kept

mind. Most stammerers probably inherit an inferior speech apparatus (often linked with a left-handed or ambidextrous tendency). This may be harmless unless the social situation and the patient's interpersonal relations precipitate anxiety and fix it into psychoneurosis. The neurosis takes hold of the least resistant system and causes symptoms. The etiology is multiple, and treatment must be varied to match it. The inheritance of a weak speech mechanism is no obligation to stammer. Emphasis in treatment should be put on amelioration not cure, for few are ever entirely relieved of symptoms.

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